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THE UNIVERSITY OF TORONTO MEDICAL JOURNAL

(An Undergraduate Publication)

This journal is published seven times a year, November to May, by the Medical Society of the University of Toronto, to serve as a medium for student expression and to bring to the student recent articles of scientific interest.

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Volume VII TORONTO, MARCH, 1930 Number 5

EDITORIAL.

This month and next we will be without the able services of our editor-in-chief and, resisting the universal temptation of the mouse to play when the cat's away, we shall carry on along the old lines. The present issue will probably have a greater proportion of staff articles than previously, as time approaches the merry month of May, we find it harder to obtain articles from the students. Oddly enough at the moment of writing this, we were called to the 'phone by a II. year student who explained that he had an unsolicited article which he wished the Journal to publish. This seems to be a realization of some of our ephemeral dreams.

Concerning the subject matter of the articles, we have two viewpoints; one, that they should be a presentation of the modern thought and research upon the subjects or diseases which, as it might be said, are the 'bread and butter of the general practitioner.' The other view is, that we should present articles upon a more theoretical or abstract subject which might be the student's desire to read up, but for which he has little time.

Of these views, the former is probably favoured the most; the physicians, amongst whom we have many subscribers, desire material which will help them in their daily work: the upper years wish articles which will aid them in passing their examinations and councils; similarly the lower years, articles dealing with their particular phases of medicine.

It has always been the intention of the Journal to present articles you de-

sire: if you will not write them, you might at least indicate what subjects you would appreciate most.

ON THE IMPORTANCE OF THE FUNDAMENTAL DIAGNOSTIC METHODS IN MEDICINE.

In order to become a decent diagnostician a certain substructure is necessary.

(1) The physician must be a reasonably good pathologist. It is futile and impossible for one to attempt to recognize by means of physical examination, structures and changes in organs with which he is so unfamiliar that he would not recognize them were they before his eyes.

(2) We must have acquired by experience, skill and confidence in the arts of ausculation and percussion. This skill and this confidence can only be obtained by correlating one's clinical observations with the anatomical changes observed after death.

(3) We must have a good training in anatomy and in the simple methods of neurological diagnosis, which require no elaborate mechanical procedure, but which do require patience and accuracy.

(4) It goes almost without saying that real skill as a diagnostician comes only through experience in the practice of the art.

Since the XIXth century the advance in pathological anatomy and physiology, the development of our knowledge and bacteriology and serology, the increasing application of physical, chemical and physiological methods to medical investigation, have greatly augmented our diagnostic resources.

The increasing uses of these methods have brought it about that more and more men and women are devoting themselves to special branches of applied science. The practitioner must delegate this branch of work to these men,—it is impossible for him to obtain the equipment or the specific knowledge. Similarly, the laboratory man, though proficient in the interpretation of his tests, has not the time to do much in the way of practice. Each man has, as it were, a blind side to his medicine.

Although our resources are greater, though we have many new procedures to help us out, there is not one which takes the place of the fundamental methods of observation, palpation percussion and auscultation. To these the new methods are all complementary. Sergent has well said: "One sense may not be substituted for another. The senses are complementary."

As far as the general practitioner is concerned, beyond certain simple routine procedures; such as, the ordinary urine examination, a glance through a microscope at a fresh specimen of blood, with perhaps a simple Haemoglobin estimation on the Tallqvist, he must know the limitation of the laboratory to which he may appeal. And always, he must consider his patient for, pleasant and relieving as it may be to appeal to every possible method of study, the patient often can ill afford to pay for that which is absolutely necessary, and it depends upon his physician to protect his pocket from unnecessary strain.

In our modern practice it is far too common to neglect or slur the simple methods of physical examination which, in the great majority of cases, are sufficient to give us a reasonable concept of the situation; and to appeal at the onset, without proper observation or examination, to the laboratory or to the Roentgenologist, and worse than that, to accept the report as diagnostic without realizing that these examinations are complementary to and should be considered only in connection with a preliminary physical examination and consideration of the general situation.

Similarly, the examination should be systematic. Preconceived ideas, the

inability to keep an open mind and to consider judiciously all available evidence before conclusion and action, have ruined many careers. The accuracy of the "snap diagnoses" of the experienced may be impressive but a "snap diagnosis" is an abomination.

There is curiously enough a certain tendency to regard as definite and final the report from a clinical laboratory. To begin with, the tests themselves are not infallible and, in addition, they are made by human beings who are, as we all are, subject to error. Roentgenology is a very great aid to diagnosis, yet all we are reading is light and shadow, there is nothing definite in it; the value depends on conditions into which the human element enters in the interpretation of these lights and shadows. To accept blindly the opinion of a Roentgenologist on the presence or absence of, let us say, tuberculosis, is a doubtful case; or his opinion in a given case in its activity or non-activity, without a full consideration of the clinical history and physical signs, is preposterous.

If we carry away the few facts which follow, your time in reading this will be well spent.

(a) There are few short cuts in diagnosis.

(b) The proper training of the student and the practitioner in the simple fundamental procedure of physical examination, is perhaps the most important function of the School of Medicine.

(c) That their conscientious employment is a necessity for him who would become a good doctor.

In the preparation of this I owe a great debt to a paper by William Thayer, M.D., "Colorado Medicine, Oct. 1929."

The Analysis of a Muscle Contraction Into Its Constituent Components.

By ALBRECHT BETHE, M.D., Ph.D.,
(Frankfort on Main).*

The normal curve of the muscle written by a lever is of great practical interest, but it tells us nothing about the internal mechanical processes in the muscle itself. This common curve is only the sum of all the mechanical processes which may be either active alone, or partly active and partly passive. If the muscle were to contract simultaneously in all its parts and if it consisted of solid substances without plasticity—then this curve would be a true expression of what is occurring. But it is not, as every one knows. Generally the contraction begins in a certain region and runs like a wave from there to the other parts. The contraction begins when you excite the muscle from the nerve in that region where the nerve ends. When you stimulate the curarised muscle directly, for instance, with a constant electric current, the contraction begins at the cathode, if you close the circuit, and at the anode, if you open it.

Furthermore, it is easy to show the muscle is a string with a rather low and incomplete elasticity. When you load the resting muscle with a small weight it is lengthened, and if you increase the weight the muscle becomes

*Albrecht Bethe, M.D., Ph. D., is Professor of Physiology at Frankfort on Main. He was invited as special lecturer to the University of Toronto in Sept., 1929. This is the first of two papers presented, the other will appear next month. Translation is by courtesy of Prof. Best and the Department of Physiology.

longer and longer. The same thing happens, but to a lesser degree, when the muscle is kept contracted by tetanic stimulation.

Consequently it was to be considered probable—as some earlier experiments indicated—that on stimulation the muscle would not merely contract, part after part, but that the parts not yet contracting would be stretched at first before they begin to shorten. In order to prove this it was necessary to record the movements of various parts of the muscle in contraction. This would be performed only by a myophotographic method. First I will explain this method.

One attaches distinct cross marks at regular distances to the muscle; these marks will show the movements which the part between two of them undergoes. One then photographs these cross-strips through a narrow slit on a quickly moving photographic paper during the contraction. But that is easier said than done, and the difficulties were greater than we thought in the beginning of our experiments.

FIGURE 1.

With nothing but the light reflected by the muscle one does not get photographs with sufficient contrasts. So we could use only transparent muscles, such as the Sartorius of the frog. Even then the light must be so bright that one can expose the muscle to the rays only for a short time, in order to prevent its becoming too hot. To get the light we wanted, we used three condenser-systems. One of them was a cylindrical lens.

After several unsuccessful attempts we used for marking the muscle black silk thread which became sufficiently firmly fixed by mere capillarity. These prepared muscles are put into a moist chamber which is placed on the optical bench where the elliptical light-field is of the greatest intensity. The photographic system projects a sharp picture on the photographic paper fixed on a drum. A screen with a long and very narrow slit is placed before the paper. Beside the slit the screen is perforated at regular distances. By these small holes the light penetrates and writes the base lines. Time-ordinates are given by increasing the light of the electric projection lamp at regular intervals. This was carried out by a tuning fork, which removed a shunt every hundredth of a second.

In this way we got photographs such as Fig. 1. Here you see the curves of a curarised Sartorius stimulated by a single induction shock which passed through the whole muscle. The cathode is fixed near the lever. Below you see the ordinary muscle-curve written by the shadows of the black silk threads. They become lower and lower as they approach the fixed end of the muscle. But you see that each of these curves differs distinctly from the lowest curve. They all begin with a depression whose upward turn occurs the later the more distant the part of the muscle is from the cathode. Here you see immediately that in the first moment of mechanical response only the directly stimulated part of the muscle is contracting; the

more distant parts are stretched at first.

This fact is only conspicuous if one stimulates the muscle at the movable end. If the muscle is excited at the fixed end, no curve will show a depression and all will be more or less similar to the curve of the free end or of the lever.

How may we explain this contraction. It only seems to be a contradiction; each curve of the photographs represents the sum of all mechanical reactions which occur in the muscle between the fixed point and the mark. In order to analyse the mechanical process in the muscle and to obtain the true relationships we have to construct new curves which represent the differences between the movements shown by the photographic curves of adjacent marks on the muscle.

In order to get these partial curves one has to magnify the original photographs and to draw each curve on the same abscissa. Then one takes the difference between two adjacent curves of this drawing and so gets a new series of curves which represent the movements of all single parts of the muscle.

Fig. 2 shows such a series of partial curves taken from the represented original photograph (Fig. 1). The curves of the parts stimulated first (7 and 6) rise immediately. All the others show at first a depression which lasts longer the greater the distance between the part concerned and the point of excitation (see the curves 5 to 1 in Fig. 2). If we excite the muscle on the fixed end we get, as already mentioned, no depressions in the original photograph, but in the redrawing they come out very well. It looks like Fig. 2, only the sequence of the curves is reversed. The curve of part 1 rises first, the curve of part 7 last. (See also Fig. 3c and d).

The ordinates of Fig. 2 indicate the height of contraction given in percentages of the length of each muscle part. You see that the contraction is every-

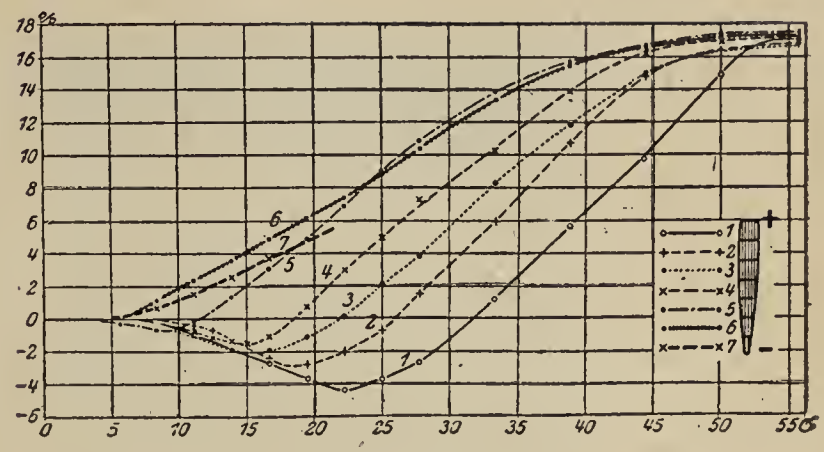

FIGURE 2.

where proportional to the length. That proves that there is no decrement of the muscle-wave. In addition we see

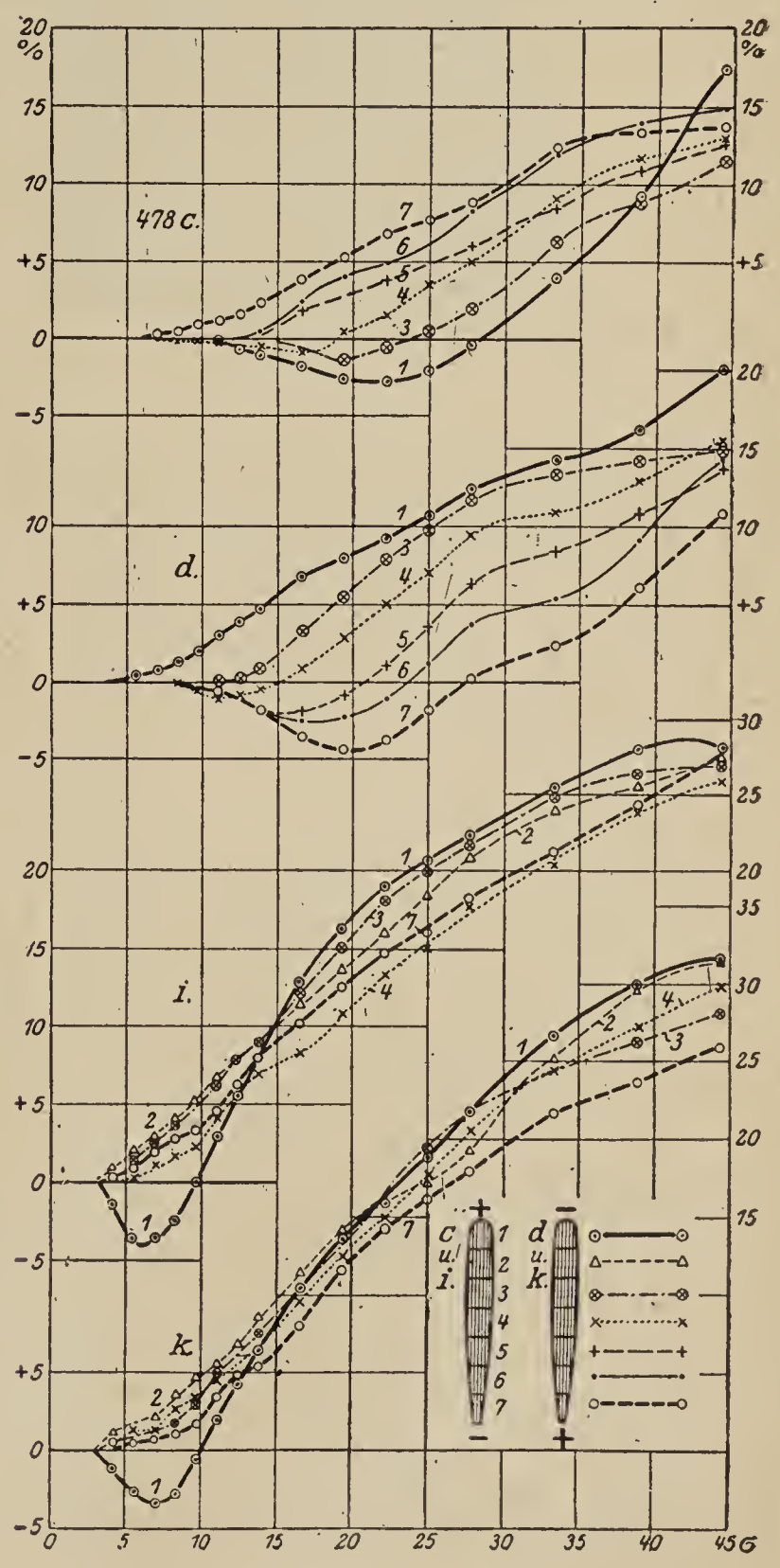

FIGURE 3.

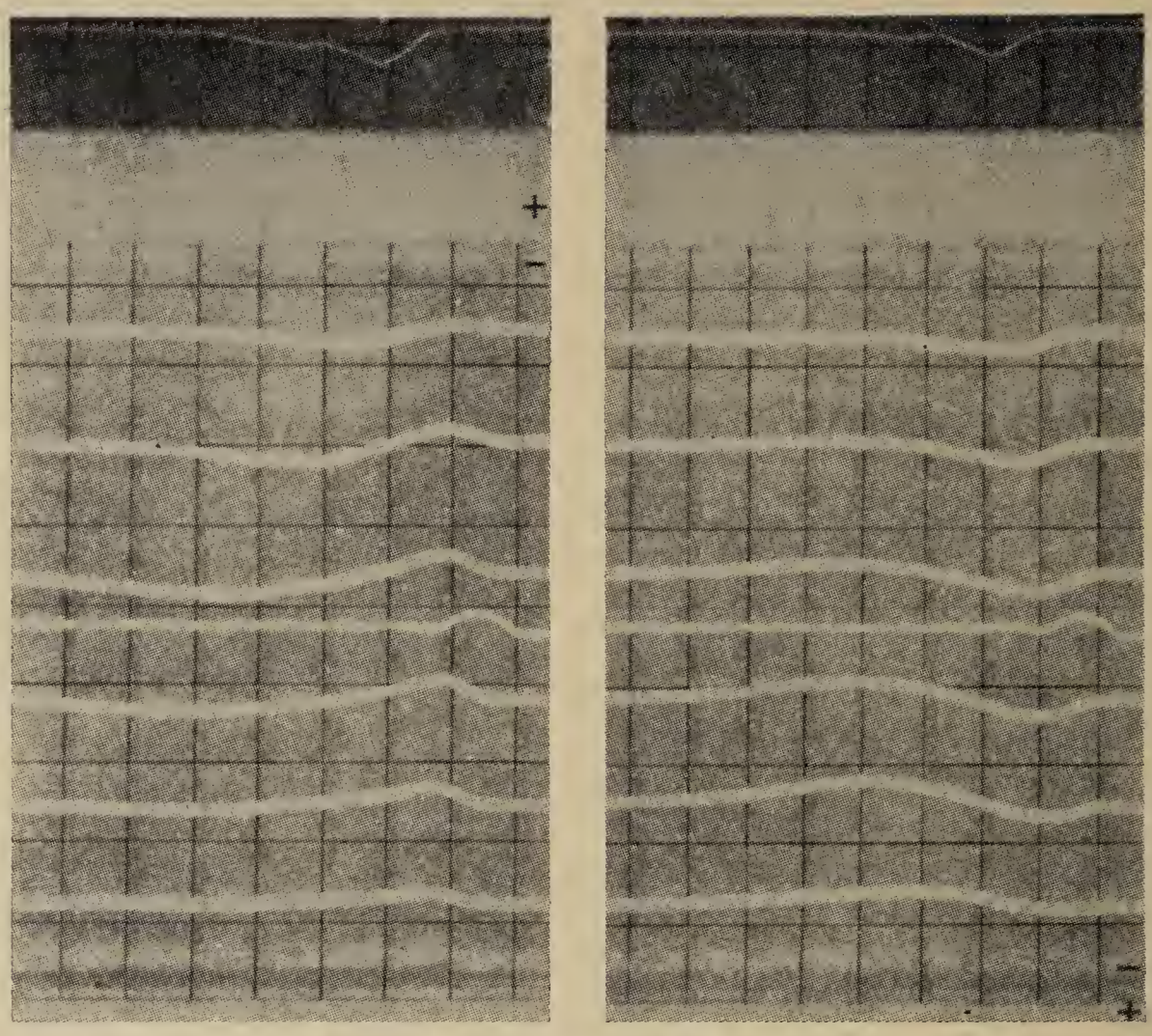

FIGURE 4.

that there is no stretching of the first two pieces near the point of excitation, and that the stretching always increases as the part of the muscle is more distant from the stimulated part. The latent period of the firstly contracting piece is less than 5 Sig.*; then it increases more and more, showing the rate of the contraction wave. Afterwards we shall come back to this fact.

The form of the original curves depends only on the situation of the cathode of the induction-shock and not on the situation of the anode. The mass of the muscle is always drawn towards that point where the stimulating cathode is lying. The situation of the anode may be anywhere. But this independence from the situation of the anode is only true if the stimulus is not too strong. If both electrodes were placed close together on one end of the curarised muscle, the same form of curves was obtained with strong stimuli as with weak ones. Only the sequence of the partial curves becomes inverse when we excite the fixed end in one case and the movable end in the other. If the stimulus is weak or medium we get the same effect by fixing one electrode on each end of the muscle (Fig. 3c and d). But if the stimulus is strong all curves begin to rise nearly at the same time, and there is no longer any difference in the sequence (Fig. 3i and k).

This result corresponds with the well known fact that the latent period of the whole muscle is much shorter after a very strong stimulus than after a weaker one—but only if the induction shock flows through the whole length of the muscle. The only explanation of this fact is that the law of polar excitement is only entirely true for weaker stimuli. When the stimulus is strong every lack of homogeneity will produce a cathode and an anode on the two sides of the heterogeneity. If, for instance, the layers of the striated muscle fibers represent such heterogeneities, then the muscle is ex-

* .001 sec.

cited in its full length at the same time.

Until now I have spoken about myophotographs taken with an isotonic lever. The photographs taken by the isometric method are much more difficult to understand. It was Dr. E. Fischer who undertook these experiments. Fig. 4 shows one of his original curves; below is the curve of the isometric lever, in the middle the signal showing the amount of excitation. In the first case he stimulated the one end of the muscle, in the other case the other one. One sees that the muscle does not keep motionless, as Hill suggested, but that its parts flow to and fro, in the first case in one, in the other case in the opposite direction. One gets nearly the same effect if one separates the electrodes and attaches them to the opposite ends of the muscle. What really happens in both cases we can distinguish only by the differential curves (Fig. 5. In all cases that part which gets the stimulus first contracts first and will be stretched a short time later. All other parts are stretched first—the time of stretching being longer the more distant they are from the spot of stimulation. Afterwards they too will contract the sooner the nearer they are to the other end. By stimulating the other end you get the same phenomena, but in opposite sequence.

Now we shall see how the muscle reacts when it is not curarised and when it is excited either directly or from the nerve by one single induction shock.

I begin with the results of the isotonic method; if both the electrodes are lying near one of the ends of the unpoisoned Sartorius, that means in nerve-free regions, then the photographs do not differ from those taken from the curarised muscle with the cathode placed on one end and the anode on the other end. But the effect upon the unpoisoned muscle is quite different when the induction shock is sent through the full length of the muscle. There is no longer any difference between the curves we get with the cathode placed on the movable end and those with the cathode on the fixed end. But still more astonishing is, that a stimulus from the nerve gives the same form of curves. Not only the

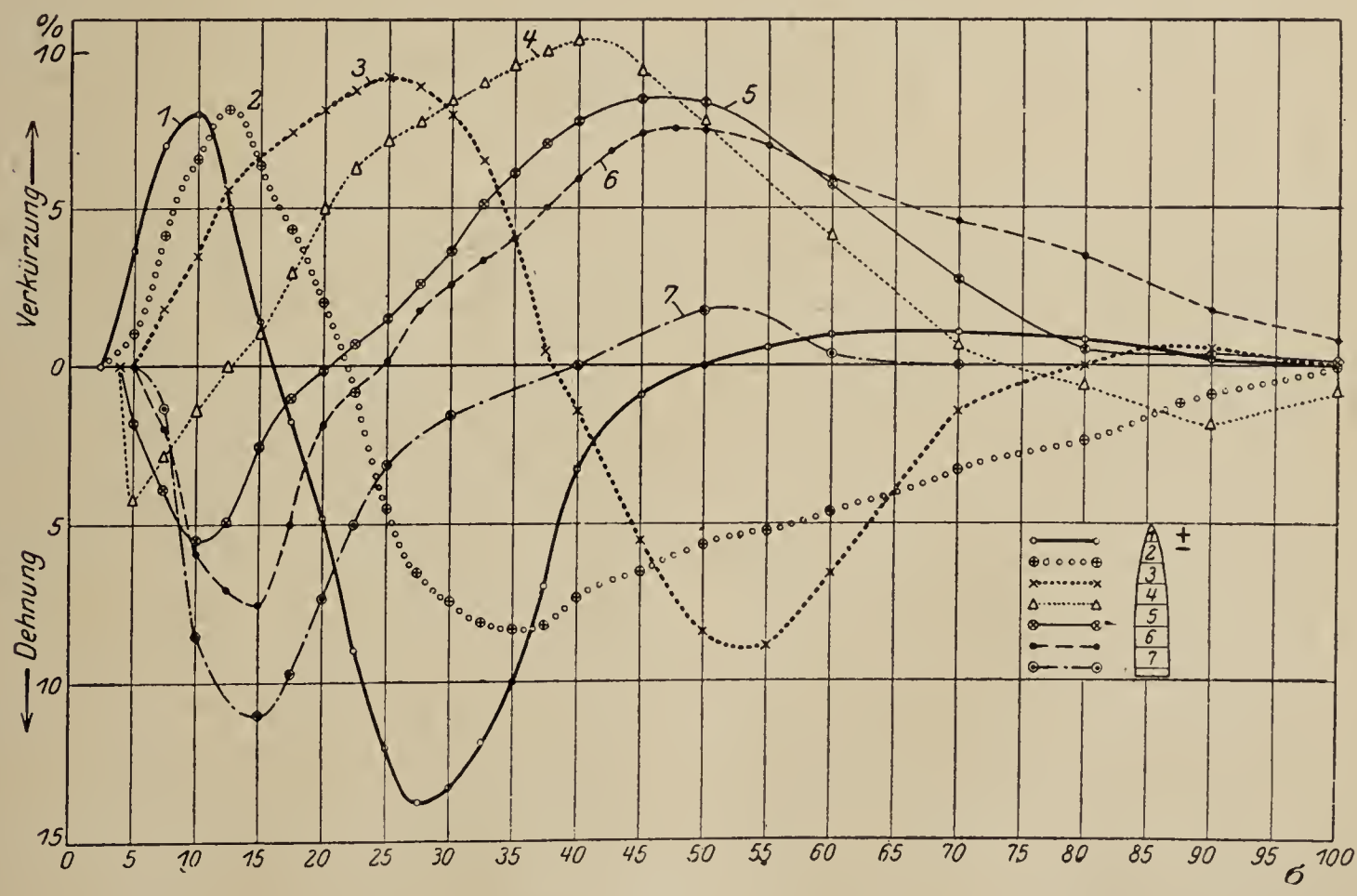

FIGURE 5.

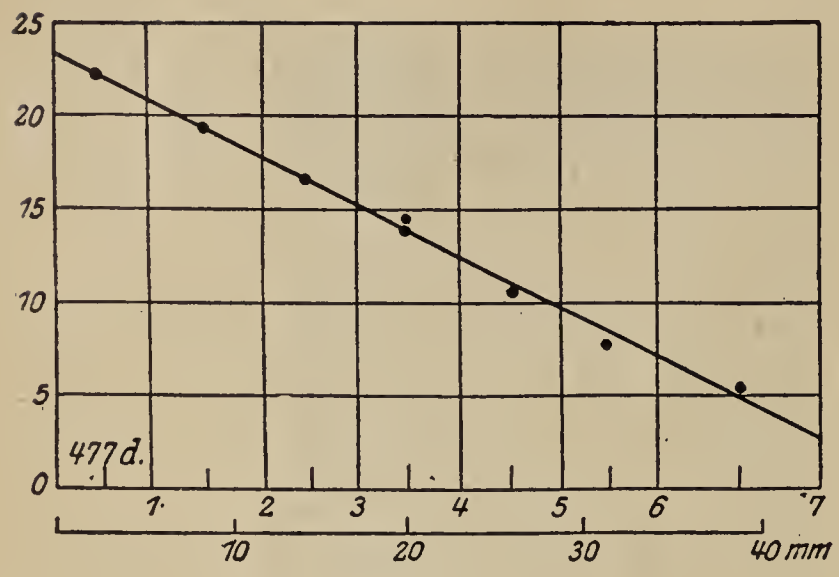

FIGURE 6.

arrangement in the series of the curves is nearly the same, but also the sequence. This proves that exciting the unpoisoned muscle by a submaximal induction shock going through the whole muscle does not excite the muscle itself, but always the nerve endings.

In Fig. 6 the abscissae show the positions of the black marks applied to curarised muscle; the ordinates represent the latent period of the corresponding muscle pieces in sig. You see that all these points lie nearly on one straight line. That means that the rate of the muscle wave is the same in all parts of the muscle. If we produce the straight line to that point which signifies the end of the muscle fibers, we still get a positive time of about 2, 5, sig. That will be the real latent period of the smallest muscle piece. It is nearly the same latent time we get directly by a very strong induction shock going through the whole muscle. We saw that such stimuli excite nearly all parts of the muscle at the same time, so that the lever begins to move nearly in the same moment as the contraction begins.

By the method we employed in our laboratory, we did not get results which could not be expected according to former experiments. But it seems to me that the mechanics of the muscle became a little clearer than they were before.

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Anaesthetics.*

By V. E. HENDERSON.

The Editor of the Bulletin has asked me to write a short article on Anaesthesia, which will be illustrated by the experimental work carried on in the Department of Pharmacology during the past seven years.

The first important study of anaesthesia arose from a suggestion of Dr. W. Easson Brown, at that time a Fellow in the Department, that some means should be found by which nitrous oxide could be a more satisfactory anaesthetic, and this leads me to discuss some of the commonly used anaesthetics from the standpoint of their disadvantages or dangers.

Nitrous oxide will not produce anaesthesia if given in lower concentrations than at least 95%. Even with 95% nitrous oxide and 5% oxygen in many patients no complete unconsciousness is produced, and in consequence, the ordinary procedure is to administer for a short period of time (1 to 2 minutes) nitrous oxide in 100% concentration. Then oxygen is gradually introduced, but it is rarely that the concentration of nitrous oxide falls below 93%. I am speaking now of ni-

*Reprinted by courtesy of University of Toronto Medical Bulletin.

trous oxide without preliminary administration of morphine and hyoscine. Even with these high concentrations of nitrous oxide, relaxation of the muscle is not as a rule sufficiently obobtained to make easy the carrying out of surgical procedures in the upper and lower abdominal cavities. It is for these purposes that very frequently nitrous oxide and oxygen is enriched by ether in low concentrations. This enables more adequate relaxation and also the administration of larger amounts of oxygen.

The real danger of nitrous oxide lies in the absence of oxygen. Almost from the first of an anaesthetic under nitrous oxide, the red blood cells carry less oxyhaemoglobin to the tissues, and the higher the concentration the lower the oxygen content of the blood falls. The consequence is that the tissues are throughout suffering from lack of oxygen, and it is not surprising that cases of cardiac failure, either fatal or dangerous, occur from time to time with this anaesthetic.

Many years ago Paul Bert suggested that this difficulty could be overcome by administering nitrous oxide under pressure in a chamber where the pressure amounted to an atmosphere and a quarter or thereabouts—he gave animals the equivalent of 100% nitrous oxide with 20% oxygen, and thought that he obtained satisfactory anaesthesia. His procedure has been little used, but for other reasons to which I will refer presently, we repeated his experiments (Drs. Brown, Lucas and myself), and found that in cats we were unable to obtain any degree of anaesthesia even at pressures up to nearly two atmospheres, unless the quantity of oxygen was reduced to about half its value in air. The fact seems to be that in nitrous oxide anaesthesia the nitrous oxide produces a certain degree of amnesia and depression of the cerebrum, but this is not adequate to produce complete surgical anaesthesia unless accompanied by a lack of oxygen. It is the lack of oxygen which is the important supplement to the presence of the nitrous oxide. In other words, nitrous oxide anaesthesia always entails a certain lack of oxygen in the tissues, and this is true even if given with morphine and hyoscine as premedication. Only this lack of oxygen has certain deleterious effects. This can be most easily observed in a change in the acid base balance of the body towards an uncompensated acidosis and in the cessation of liver function which often persists for 12-18 hours. It was these concentrates that led us to seek a new anaesthetic more potent than nitrous oxide, but, like it, gaseous in form.

Some suggestions in the scientific literature led us to examine the action of ethylene as an anaesthetic in some detail, and we found that it produced anaesthesia in cats when given in a mixture of 80% ethylene and 20% oxygen. In practical human anaesthesia a higher concentration of ethylene is usual 90-95%—for a short time, and if great relaxation is required it is often impossible to give more than 10-12% of oxygen throughout the operation. This is better than with nitrous oxide, and it is found that even under these conditions the upset of the acid base equilibrium is less than with nitrous oxide, and in cases where adequate oxygen may be given (that is when the deep third stage of anaesthesia is not required) there is but slight metabolic change. The effects on the liver and the movements of the intestines disappear as quickly as with nitrous oxide, if not a little more rapidly. Like nitrous oxide, there may be vomiting during the first hour, but this rarely persists.

Our scientific knowledge led us to believe that propylene, a higher member of this same series, would be a more potent anaesthetic. It was studied and, as was expected, it was found that it produces anaesthesia in the mixture of 65% propylene with 35% oxygen, and that long periods of anaesthesia

could be carried out with but little change in metabolism. Unfortunately when propylene was produced and compressed for human administration, it was found that it differed from that made in the laboratory in that it caused an undue secretion of bronchial mucus and a lowering of the blood pressure which did not occur before. This has led to its not being used in human anaesthesia on any scale, and until the peculiar chemical change which takes place has been elucidated, it is not likely to come into favour.

In searching for the source of this toxicity, Dr. Lucas was led to think of cyclopropane. On investigation it proved to be not a toxic substance, but an anaesthetic of remarkable potency. Some 12% produces anaesthesia. In deep stages of anaesthesia, blood pressure remains high. Even in the presence of 30% instead of 12%, blood pressure may remain within normal ranges (the respiration is slowed and less ample). Nevertheless, there is little or no upset in metabolism. Unfortunately, at the present moment, the materials from which the gas is prepared are expensive, and unless it is manufactured with great care it is apt to contain traces of an impurity which resembles in its effects that found in tanked propylene.

It has long been known that both ether and chloroform produce a very great upset in acid base metabolism, and at the end of a period of anaesthesia of half an hour or longer, there is a well-marked acidosis and that the functions of the liver are depressed and remain so for many hours. With ether, the liver has almost completely recovered in 24 hours, with chloroform the recovery may not be complete for more than three days.

An examination which has been carried on for us by Dr. J. E. Bates in the Department of Pathology, of the livers of animals who have been anaesthetized for long periods of time with these anaesthetics, has shown that at the end of 12 or 14 hours there are slight pathological changes in the liver with both ether and chloroform, but at the end of 24 hours the ether livers appear to have recovered while the chloroform livers show more marked intoxication. It is known that in some cases of chloroform administration, especially if prolonged, there is a subsequent fatty degeneration of the liver which may prove fatal. This appears to occur more often in emaciated individuals. It has long been suspected that this degenerative change might be due to the breaking up in the body of a small amount of chloroform. Owing to the large amounts of chlorides normally present in the body, one could hardly hope to detect the breaking up of even 2% of the chloroform inhaled. However, by administering bromoform, which has the same chemical structure as chloroform, save that bromine has replaced chlorine in the molecule, Dr. Lucas was able to show that in animals as much as 2% of the bromoform administered might be broken down and be excreted as bromides, and that the bromides could be found in the urine for days and also in many tissues of the body.

Bromoform causes the same pathological changes in the liver as chloroform. We were further able to show that macerated liver pulp broke down chloroform at body temperature. Other anaesthetics, such as ethyl bromide, are also in part broken down in the body, and there seems little doubt that some of the constant actions of this group of anaesthetics which contain bromine or chlorine, are due to their being broken down into toxic products within the cells of the body.

The reader will have noticed that I have indicated two sources of toxicity. One type is illustrated by chloroform where some of the chloroform undergoes chemical change within the body, giving rise to toxic products. This, we know, is one of the causes of toxicity of methyl alchol, as compared with

ethyl alchol, as methyl alcohol is oxidized in part to formic acid.

The second type of toxicity is that illustrated by nitrous oxide, and to a certain extent ethylene, which depends upon its interfering with normal oxidations in the body, owing to the high concentration in which the anaesthetic gas must be given. It seems most probable that the metabolic changes produced by ether are not of this type, but are due to some of it being split up within the body. The most careful experimenters have not been able to regain more than 90%-95% of this absorbed by animals or patients, in the expired air, urine or other excretions. Nitrous oxide, and probably ethylene, propylene and cyclopropane, are exhaled intact without change.

There is, however, a third type of toxicity which seems to be inherent in the anaesthetic, and this usually shows itself in its effects on the central nervous system, or possibly on the heart. It is well known that ether tends to stimulate respiration at first, while chloroform depresses it. With ether, the blood pressure falls slightly or not at all, while with chloroform there is a steady and progressive fall of pressure, owing to its effect on the vaso motor centre. Chloroform, too, is obviously more depressing to the spinal cord than is ether, and both of these appear to be more depressing than are the gaseous anaesthetics of which I have spoken.

A peculiar example of this inherent type of toxicity was revealed by the study of butylene. This gas produces anaesthesia in about 40%, but almost as rapidly as the animal becomes anaesthetized, the blood pressure falls to quite low levels and recovers as promptly when the anaesthetic is removed. This again was due to its exerting some specific effect on the vaso motor centre. It also tended to produce a very marked rigidity or spasm of the muscles of the limbs, which lasted to a deep stage of anaesthesia.

This leads me to call attention to the fact that sometimes this rigidity or increase of tonus in the muscles of the extremities, is seen from time to time with ether and earlier with chloroform. In some of the cases with ether this has left the arms and legs showing very marked clonic movements. Here again, we have a type of disturbance which seems to be inherent in the anaesthetic.

Undoubtedly the safest anaesthetic for general employment in the hands of the non-expert is ether; very much less safe for prolonged anaesthesia is chloroform. For the patient who has been badly shocked surgically, nitrous oxide is undoubtedly better than either of the others, as the war experience amply showed, but it must be administered by one who is thoroughly familiar with its dangers and who realizes that if given without preliminary medication, it entails a serious strain on the patient. Given with preliminary medication of morphine and hyoscine, it is much safer, but again entails a greater degree of expert knowledge. Ethylene appears to be safer than nitrous oxide, but has the great disadvantage, as has propylene, acetylene and cyclopropane, of being explosive. This danger should be thoroughly understood by any anaesthetist. Like ether, it cannot be used in the presence of an electric spark or an open flame. The only fatal cases have been where these precautions were neglected. Minor cases of explosion have occured owing to the static charge developing in the machine and possibly present on anaesthetist and patient, and which resembles the spark that can be obtained by shuffling on the carpet on a frosty day, and touching a radiator. This can certainly be avoided by relatively simple means. Similar explosions have occured, I have been informed. with nitrous oxide and oxygen enriched with ether, and indeed the danger of explosion is quite as great with this procedure as with ethylene.

Ethyl chloride is a dangerous anaesthetic, but in my opinion almost essential in the armamentarium of the expert anaesthetist for short anaesthetics in 1 or 2 minutes in suitable subjects. Unfortunately anaesthetists have grown careless and forgotten its dangers. If it is used for induction, the mask should be changed before proceeding with ether, nor should the mask be so saturated with ether, as to prevent the exhalation in the first few minutes of the ethyl chloride absorbed by the patient. And further, the change should be made at a very early stage.

I cannot close without calling attention to the very great advantages occuring from the administration of carbon dioxide in about 5% concentration during an anaesthetic to maintain ample depths of respiration, and the consequent opening up of as many lung alveolae as possible, with the result of conveying ample amounts of oxygen to the tissues, and particularly to its great advantage after anaesthesia for a short period in order to rapidly evacuate the ether from the body, and also to open up the small bronchioles which may have tended to become clogged by mucus during a period of shallow breathing. I have no doubt that this procedure is going to greatly decrease the number of cases of so-called 'post operative pneumonias.'

Thyroid Storms.

By C. A. WICKS, B.A., 3T0.

"Thyroid storm" is the descriptive phrase used to designate the clinical picture presented by a hyperthyroid patient who is passing through an acute exacerbation of his hyperthyroidism. Such terms as thyroid crisis, thyroid delirium and acute hyperthyroidism are used by various authors in referring to essentially the same condition.

With the clinical and pathological manifestations of typical hyperthyroidism, we are familiar. It will suffice merely to mention the several types,—

(1) Diffuse hyperplastic goitre. (i. e.) Exophthalmic goitre.
(2) Adenomatous goitre with hyperthyroidism. (i. e.) Toxic adenoma.
(3) Combined toxic adenoma and diffuse hyperplasia.
(4) Iodine thyrotoxicosis.

INCIDENCE.

There is a complete absence of reliable statistics in the literature regarding the incidence of thyroid storms at the various clinics. There is universal agreement, however, on the fact that such crises have been reduced in frequency and severity in a very spectacular manner during recent years. This reduction has been made possible by,—

(1) A greater number of patients receiving adequate treatment in the early stages of the disease.
(2) Great advances in the surgical management of patients with hyperthyroidism.

Acute hyperthyroidism may arise at any time prior to operation, during the course of the operation or immediately following operation. The preoperative storms are almost entirely confined to the diffuse hyperplastic goitres. Operative and post-operative storms are encountered in all types of hyperthyroidism.

While the proper management of these patients has almost eliminated the severe storms, moderate reactions are quite common. Furthermore, the importance of the severe crises, when they do occur, may be judged from the following statement by Pemberton [16].

He lists the three outstanding factors in the surgical mortality of hyperthyroidism as,—

(1) Surgical accidents (hemorrhage, nerve injury, etc.).
(2) Acute post-operative hyperthyroidism.
(3) Myocardial and vascular damage (usually the result of late treatment).

CLINICAL PICTURE.

A thyroid storm may approach suddenly and rapidly attain severe violence, or it may gradually build up in intensity, being heralded by significant signs. They may be of all severity from a simple bout of irritability and restlessness to the most profound toxaemia which, in spite of energetic treatment, will terminate fatally.

The signs of an approaching storm are,—(1) increased pulse rate, (2) increased restlessness, (3) increased irritability, (4) lessened emotional control. The pulse may reach a rate of 180 to 200. Dyspnea is often a prominent symptom. An occasional patient complains of epigastric pain. The restlessness is evidenced by the threshing about in bed, purposeful but useless movements and rapid inarticulate speech. The least disturbance arouses a stream of comments and a mental reaction which still further agitates the hypersensitive mental balance. As the above symptoms increase in severity, the patient takes on an irrational attitude which, as Plummer says, may resemble that of a drunken soldier. Delusions are common. The face is flushed. Sweating may be profuse.

The actual crisis is usually ushered in by an attack of diarrhea or vomiting. From this point the patient rapidly becomes worse. The temperature soars to 104 or 105 degrees. Delirium is now present and mechanical restraint may be necessary to protect the patient from injury. Respirations increase in rate, the pulse continues to rise and auricular fibrillation frequently occurs with a marked pulse deficit.

As the case progresses, the vomiting and diarrhea usually become less frequent, delirium alternates with periods of apathy or stupor and the sweating is replaced by a dry skin. The temperature mounts to still further heights. Acetone may be detected on the breath. Coma sets in and terminates in death.

An impending crisis on the operating table will be recognized by the anaesthetist. Danger signs under these circumstances are,—(1) a persistently high and rising pulse rate, (2) a steadily rising systolic blood pressure especially when associated with a lowered diastolic pressure, (3) rapid respirations.

The reaction to operation may be so acute that the patient is in a serious condition on returning to bed. The obvious signs of such post-operative crises are, a very fast pulse, extremely high temperature, great restlessness, profuse perspiration and mumbling speech with delirium.

PATHOLOGICAL CHEMISTRY.

(1) High metabolic rate. Obviously, the taking of a basal metabolic rate at the height of a storm is impractical, and therefore we have no accurate measurements as to how high this metabolism may reach. If the pulse rate or the temperature be any indication of the work being expended by the body tissues, we may certainly assume that catabolism is extremely rapid. The determination of the nitrogen of the urine under these circumstances should be productive of interesting results.

(2) Acidosis. Before the storm overtakes the patient with hyperthyroidism the glycogen reserve of the liver is below normal. This will be so unless he is taking sufficient carbohydrate to supply the fuel for his increased metabolism. The victim of a storm will require still larger amounts of carbohydrate and unless this is furnished, the

glycogen reserve will be depleted. Acidosis will be the inevitable outcome in such a case. On top of this glycogen deficiency, there is possibly superimposed a deficinecy of available insulin which will still further reduce glucose oxidation. It is well known that experiments demonstrate the antagonistic action of the internal secretion of the thyroid upon that of the pancreas. Furthermore, Jackson has observed several cases of hyperthyroidism associated with true diabetes mellitus which latter condition markedly improved following the surgical treatmen of the goitre.

(3) Toxaemia. The toxic element in exophthalmic goitre is believed to be a poorly iodized thyroxin (i.e.) qualitatively abnormal. In toxic adenoma, the toxic factor is more difficult to explain, but that such exists is suggested by the associated myocardial damage with hypertrophy. The diarrhea and vomiting observed in the crisis of exophthalmic goitre are evidences of the extreme toxicity.

(4) Dehydration. As a result of the diarrhea, the profuse perspiration and the vomiting in conjunction with the lack of fluid intake in an untreated case, the victim of a thyroid crisis rapidly becomes seriously dehydrated. As in a viscious circle, this condition now tends to magnify the effects of the toxaemia and acidosis.

(5) Hyperpyrexia. The heat dissipating mechanism is seriously disturbed when dehydration sets in, so that body temperature rises still higher. Temperatures per axilla of 107 to 108 degrees have been recorded in severe thyroid storms. This will further increase the metabolic activity by increasing the rate of intra-cellular reactions.

The heart is labouring with its weakened myocardium to provide oxygenated blood to tissues which are being stimulated to excessive activity.

The hyperexcitable central nervous system finally gives way to the abnormal products of metabolism, and coma results.

Summing up therefore, in a severe thyroid storm we have the following conditions,—(1) tremendous catabolic activity, (2) acidosis, (3) toxaemia, (4) dehydration, (5) hyperpyrexia, (6) a labouring myacardium, (7) an untable nervous system.

ETIOLOGY.

Some of the common factors which have been responsible for the development of thyroid crises are as follows,—

(1) Excitement, worry or great mental activity.
(2) Extreme physical fatigue.
(3) Intense emotional stimulation. Any incident which produces fright, sorrow or anger, etc., may precipitate a crisis in a severely toxic hyperthyroid patient.
(4) Infection. Foci of infection are not infrequently found in patients presenting signs of an impending storm. In one case, the incision into and drainage of a tonsilar abscess under local anaesthesia did much to abort a crisis. A persistent sinusitis will often retard the pre-operative improvement and may determine a rather severe post-operative reaction.
(5) Onset of a menstrual period, pregnancy or the menopause.
(6) Spontaneous. Plummer believes this feature of rise and fall in the severity of symptoms to be typical of exophthalmic goitre. Occasionally a fulminating case will arise suddenly and rapidly progress to a crisis.
(7) Improper use of iodine. It is usual to obtain marked improvement of symptoms in exophthalmic goitre after a short period of iodine therapy. Now, if the iodine be discontinued and the patients allowed to carry on, many will rapidly become worse, some of them reaching a crisis in two to three weeks.

(8) Operations on patients with undiagnosed or underestimated hyperthyroidism. It is recorded that patients with severe hyperthyroidism, having nausea, vomiting and some epigastric pain have been subjected to operation for an acute abdominal condition due to an error in diagnosis. Serious thyroid crises will be the inevitable result of such practices.

Percy [18] strongly advises against performing tonsilectomies in children with even mild hyperthyroidism.

In the event that two operations (one of them being a thyroidectomy) are indicated in any hyperthyroid patient, very careful consideration should be exercised as to the priority of each procedure. Bartlett [12] has discussed this question in a very practical manner in his treatise on goitre.

(9) Improperly-timed surgical interference. Even with excellent preoperative care and skillful surgical technique, post-operative storms occasionally occur. These are due, according to Lahey, in a large percentage of cases, to a mistake in the estimation of the patients' ability to withsand operation.

(10) Neglect in attention to operative details.
- (a) improper protection of the patient from the psychic stimuli of the necessary preparations.
- (b) lack of complete nerve block.
- (c) prolonged or deep anaesthesia by inhalation anaesthetics.
- (d) prolonged or excessive manipulation of the gland.
- (e) imperfect haemostasis.

(11) Absorption of toxic serum from the operative wound. Formerly thought to be important in the production of the severe post-operative hyperthyroidism, this factor is now believed to play only a small part in this respect.

In simply stating the above conditions which may lead to thyroid storms, it is recognized that we are not in every case, revealing the ultimate etiological factor or even the method of operation of those mentioned. Such a state must persist however, as long as our knowledge of the intimate nature and primary etiology of hyperthroidism remains as incomplete as it is at present.

An attempt will be made nevertheless, to suggest several mechanisms by which a thyroid storm might be set in motion.

Cannon has shown that increased secretion of the adrenals produces increased activity of the thyroid in a normal animal. Goetsch [22] has demonstrated that in hyperthyroidism the individual is extremely sensitive to very small amounts of adrenalin. Crile[10] suggests that thyroxin sensitizes the tissues to adrenalin. He says, "Adrenalism increases hyperthyroidism, hyperthyroidism increases adrenalism (i.e.) hyperthyroidism and adrenalism co-exist, each augmenting the other."

It is well known that extreme excitement and emotion are accompanied by an increased secretion of adrenalin. Therefore it is suggested that, in the hyperthyroid patient, such conditions may arouse a thyroid storm by means of their effect on the adrenals.

The intense fear experienced by many of these patients when faced with operation may account in part, by the above mechanism for the acute operative or post-operative hyperthyroidism. Playing also a large part in the production of this latter condition is undoubtedly the stimulation of the thyroid to increased activity by manipulations. It is quite probable that excessive handling of the hyperplastic tissue at operation, increases the secretion of thyroid products, liberating relatively large amounts of thyroxin or its poorly-iodized compound into the blood stream.

PREVENTION.

The problem of the prevention of thyroid storms involves the consideration of two fundamental factors.

(1) Prevention of hyperthyroidism.
(2) Given a patient with the disease, to so manage the treatment that thyroid storms do not arise.

(1) Prevention of hyperthyroidism. Since these crises occur only in patients with hyperthyroidism, then it would seem logical as the first step, to advocate measures which would tend to reduce the frequency of the disease. Here, our efforts are limited by the incomplete knowledge of the exact etiology. We may attack the problem however in at least two well established directions,—

(a) By the early surgical removal of all simple thyroid adenomas in adults, we will prevent the hyperthyroidism which would develop in approximately twenty-five per cent. of these patients.
(b) By discouraging the indiscriminate administration of iodine to adults with diffuse or nodular goitres, we will prevent the development of iodine hyperthyroidism.

(2) Given a patient with the disease to so manage the treatment that thyroid storms do not arise. It is not intended in this paper to enter into the discussion of the relative merits of medical, radiation, and surgical treatments of hyperthyroidism. Lahey [1] claims that practically no thyroid crises have occured in his experience except in those cases in which the disease had been present for some time, and treatment not sought or unsuccessfully managed. Therefore he considers the fundamental feature in the prevention of thyroid storms, to be the early and satisfactory control of the hyperthyroidism by surgery.

Balfour [21] says, "Judgment as to whether or not to operate, when to operate, just what to do, the proper pre- and post-operative care and the mastery of a difficult technique, are some of the major problems to be considered in the surgical treatment of exophthalmic goitre."

Several of these problems will now be considered briefly or to the extent in which they bear upon the prevention of thyroid crises. They are—

I. Proper pre-operative care.
II. Choice of the optimum time for operation.
III. Suiting the operative procedure to the condition of the patient.
IV. Adequate treatment immediately prior to operation.
V. Careful attention to operative details.
VI. Proper post-operative care.

I. Proper Pre-Operative Care.

The object of this period of treatment is essentially to bring the patient into a condition in which he can undergo operation with a minimum of danger. For our purpose, in this discussion, the danger will be primarily that of a severe operative or post-operative thyroid storm. The following measures are generally employed and are directed toward—(a) reducing thyroid activity, (b) detoxicating the abnormal thyroid secretion, (c) restoring a condition of good nutrition.

(1) Mental and physical rest.
(2) Quiet and pleasant environment.
(3) Psychotherapy.
(4) Large fluid intake.
(5) High caloric, high carbohydrate diet.
(6) Sedatives to ensure abundant rest.
(7) Lugol's iodine. The importance of this measure indicates further consideration.

The administration of Lugol's iodine to patients with hyperthyroidism in preparation for operation has revolutionized the whole aspect of the surgical treatment of this disease. It has contributed more than any one other

factor in the prevention and control of thyroid storms. The introduction of pre-operative iodine therapy by Plummer in 1922 has resulted as follows,—

(a) the pre-operative period has been greatly shortened,
(b) the multiple-stage operations have been rendered unnecessary in the large majority of cases,
(c) the dangerous post-operative reactions have practically disappeared.
(d) the operative mortality has been reduced from 3.5 per cent to less than 0.7 per cent.

The dosage of iodine will depend upon the degree of toxicity exhibited by the individual patient. Ten minims of Lugol's solution three times a day would appear to be an average dose. Maximum improvement with the above measures occurs rather consistently between the sixth to the fourteenth day.

It was formerly thought that patients with toxic adenoma could not be given iodine with safety. Experience has proved, however, that given over a short period of time in small amounts, iodine will often produce improvement. Coller and Porter[20] state that iodine caused a favourable effect in 46 per cent. of their patients with toxic adenomas as compared with 88 per cent. of those with exophthalmic goitres.

II. Choice of Optimum Time For Operation.

There is probably no single index of the optimum time for operation. No laboratory or clinical test takes the place of the judgment of the surgeon, experienced in the management of such patients. A low reading on a single basal metabolic test is by no means a guarantee of a smooth operative and post-operative course. Lahey says, "No operative procedures should be undertaken on any thyrotoxic patient until all details of past history and pre-operative record have been carefully considered by surgeon, assistant and anaesthetist."

The best protection against a severe post-operative reaction, is the postponement of one stage thyroidectomy until the patient (1) is gaining in weight, (2) is entirely free from apprehension; (3) shows a steadily declining basal metabolic rate; (4) has a basal metabolism below 140. Definite contraindications to any form of surgical interference are as follows:

(1) patient in a crisis or showing signs of an impending crisis,
(2) patient losing weight,
(3) emotional instability or intense fear of operation.
(4) a rising metabolic rate.

III. Suiting the Operative Procedure To The Condition Of The Patient.

Formerly, the proper adjustment of the severity of operative procedures to the condition of the patient, was one of the chief methods in the prevention of the serious post-operative reactions in exophthalmic goitre. This usually involved a polar ligation as the first step, to be followed by one or more partial thyroidectomy operations. It was essentially a process of detoxication by stages. Pemberton gives an interesting summary of the types of thyroid operation performed at the Mayo clinic for diffuse hyperplastic goitre before and after the introduction of iodine.

	Percentage of patients. Before 1922.	1927-1928
Polar ligation	70%	.6%
Two stage thyroidectomy	30%	.5%
One stage thyroidectomy	0%	ap. 99%

The above figures indicate that we are now able to bring the vast majority of exophthalmic goitre patients into such a condition that they can safely tolerate the extensive subtotal resection in one stage.

Nevertheless, definite indications for multiple-stage operations are occasionally present. Such indications are as follows,—

(1) Lack of satisfactory improvement under pre-operative treatment. That is to say, the patient has a stationary basal metabolic rate above 140,

remains nervous or apprehensive, and is not gaining in weight. These patients are usually those who have had iodine therapy for some time previous to admission to hospital and are refractory to the pre-operative course of iodine. They are said to possess "trained" thyroid glands.

(2) Appearance of a sharp reaction during operation. If the removal of one lobe of the thyroid results in even a moderate reaction it is exceedingly unwise to attempt the removal of the second lobe at that time.

(3) Evidence of extensive myocardial damage.

Of great importance is the rigid adherence to those limitations of procedure decided upon previous to operation. Having decided, on account of some indication such as those mentioned above, to perform merely a hemi-thyroidectomy, the surgeon should not be let into the mistake of attempting to complete the resection at that time, even if, following the removal of the one lobe, the condition of the patient would appear to be excellent.

IV. Adequate Treatment Immediately Prior To Operation.

Our object at this stage is to protect the patient from the psychic stimuli incident to the preparations for operation. The method by which this protection is attained varies in the different clinics. We are familiar with the "stealing the thyroid" technique developed by Crile. The actual procedure, however, must be adapted to the peculiar characteristics presented by the individual patient. Successful pre-operative treatment of hyperthyroid patients reduces their abnormal tendency to experience fear and their excessive reaction to that emotion. Therefore at the present time, elaborate measures of deception are not usually required. Mason [2] advises that the patient be brought to the operating room in a semi-somnolent state induced by premedication with morphine and hyoscine

Anticipating, and with a view to combating the increased toxicity, Eberts [9] doubles the iodine dosage on the day before and on the morning of operation.

Jackson suggests passing a duodenal tube on the evening before operation, thus facilitating the administration of food, fluid and iodine during the critical period to follow.

V. Careful Attention To Operative Details.

(1) Local block anaesthesia. Crile and others have shown that nerve block protects the central nervous system from "shock impulses" which come through even under full surgical anaesthesia. The special application of this procedure to patients with hyperthyroidism will be obvious when one considers the hyperexcitability of the nervous system in this condition. Due to the extreme sensitivity of the hyperthyroid patient to adrenalin, as pointed out earlier in this paper, this substance should not be added to the novocaine used for infiltration.

(2) Avoiding deep inhalation anaesthesia. There are numerous obvious objections to the induction of surgical anaesthesia by inhalation anaesthetics in goitre surgery. Certain objections are especially applicable to the surgery of hyperthyriodism. In this disease, due to the increased metabolism, each cell is demanding much more oxygen than it should normally require. Therefore, the disturbance in the internal respiration produced by high concentrations of such anaesthetics as ether in the normal individual, will be exaggerated in the hyperthyroid patient. Crile claims that as a result of this "sub-oxidation," symptoms of an acute thyroid reaction are initiated. [10]

Further objections to deep general anaesthesia are,—(a) The initial stage of excitement, and (b) The tendency to produce acidosis. Else [3] states

(without explanation) that this acidosis may initiate a crisis.

At the present time, most clinics are using nitrous oxide or ethylene with oxygen, in such concentrations as to produce merely an analgesia during the local infiltration or at any stage of the operation when it is indicated. The patient is in a so-called "dream state" induced by adequate premedication.

(3) Shortening the operative time.

(4) Gentleness in manipulations.

(5) Hemostasis.

(6) Avoiding all disturbing influences which would tend to arouse the patient from his "dream state" and cause any excitement.

VI. Proper Post-Operative Care.

Merely an outline of the immediate post-operative measures as they tend to prevent severe reactions, will be given here.

(1) Sedatives. Morphine, pantopon or bromides should be given in doses as required to control pain and restlessness. Kaspar maintains the patient in "twilight sleep" for two days following operation.

(2) Iodine. On return from the operating room 30 m. of Lugol's solution in saline may be given per rectum. From that time, the iodine is given by mouth in doses suited to the degree of post-operative reaction. A usual dosage is about 50 m. during the first day. This is gradually reduced as indicated.

(3) Fluids. On return from the operating room, an interstitial administration of 1000 c.c. saline with 5 per cent. glucose is given. It is essential that fluids be given to the extent of 3000 c.c. daily.

(4) Glucose. This is at first incorporated in the interstitial saline and later carbohydrates by mouth are urged.

(5) Ice caps to head and precordium.

(6) A chart of temperature, pulse and respirations recorded every four hours will serve to give timely warning of an approaching storm.

(7) No visitors should be allowed for several days after operation and the patient is encouraged to sleep by eliminating all disturbing factors.

TREATMENT.

Prophylaxis in this as well as other complications is to be preferred to actual treatment, yet crises will occasionally occur quite unexpectedly.

Lahey recognizes an impending or threatening crisis, stating that it should be regarded as an emergency and should receive prompt and adequate treatment, just as we institute emergency measures in a case of impending diabetic coma. There is definitely no emergency thyroid operation for acute hyperthyroidism. Unless the condition can be corrected by medical treatment it is futile to attempt surgery, since experience has shown that the operative mortality under these circumstances is practically 100 per cent.

(1) Remove the cause where possible. If the hyperthyroid reaction threatens to become serious at any time during the operation, Crile advises that all operative procedures should cease, the wound packed, the patient returned to bed and treatment commenced immediately.

(2) Sedatives. In an effort to control the extreme restlessness, sedatives such as morphine or paraldehyde are given. The amounts required may be much in excess of those used in any other condition. One must accept the risk of morphine poisoning as less than the effects of continued over-activation in these acutely hyperthyroid patients. Intravenous administration of sodium amytol has recently been suggested as an effective means of producing hypnosis in severe thyroid storms.

(3) Fluids. These may be given by the easiest and most effective route available. Intravenous saline administered continuously is the most dependable agent in combating dehydration

and reducing the toxaemia. Lahey advises that fluids be given slowly by the intravenous method, especially to those patients showing myocardial distress.

(4) Glucose. Acidosis will be corrected most rapidly by giving glucose with the intravenous saline in 10 per cent. concentration. Else [3] administers 15 units of insulin at the beginning and the same amount at the end of each intravenous injection of 1000 cc. of 10 per cent. glucose in saline.

(5) Iodine. The efficacy of iodine in a thyroid storm will depend upon,—(a) the type of hyperthyroidism present; (b) the question of previous iodine medication; and (c) the degree to which the storm has progressed.

It is in the crisis of diffuse hyperplastic goitre that iodine appears to exert its striking beneficial action. In the reactions following operations for true toxic adenoma it is doubtful whether iodine administration is of great value. Fortunately, however, the post-operative reactions in the latter disease are not as frequent or as severe as those experienced in exophthalmic goitre.

Improvement under iodine therapy once achieved, seldom can be repeated with the same efficacy. Therefore, those patients who have unfortunately received extensive iodine treatment previously, will be robbed of perhaps a life-saving measure when they present themselves for treatment in a crisis.

In the severe post-operative reactions of exophthalmic goitre, iodine is the most valuable and effective measure we possess, but the results are not perhaps as striking as those obtained in the pre-operative period. This observation may be explained by the fact that the patients have already reached the plateau in their improvement under iodine, before the operation was undertaken.

In the late stages of a thyroid storm we have a general metabolic disturbance affecting not only the thyroid function but all the systems of the body. Therefore it will be seen that simply correcting the dysthyroidism by iodine administration, will be solving only one aspect of the problem. To be of greatest value, then, Lugol's iodine should be given promptly and in adequate doses somewhat as follows: (a) per os—Lugol's m.xx. q. 1 h. x 5; (b) interstitially—Lugol's m. xxx. in 1000 c.c. saline x 3; (c) intravenously —5 c.c. of 10 per cent. sodium iodide in saline.

(6) Ice Pack. Eberts[9] gives detailed instructions as to the preparation and application of this therapeutic device which is indicated when the temperature threatens to rise above 103°. He claims that it relieves restlessness, lowers the temperature, pulse and metabolic rates and induces sleep. It should be given twice daily for periods of ten minutes.

(7) Removing the clips and packing the wound. Bartlett [12] has advised laying open the operative wound and packing with guaze soaked in glycerine, whenever the patient is threatened with a severe post-operative storm.

(8) Cardiac stimulants. When the pulse volume begins to fail, intravenous saline with glucose will be given with greatest benefit. Out of the mass of controversy over the advisability of giving digitalis in hyperthyroidism, emerges the fact that most writers agree on its use whenever auricular fibrillation occurs. Eberts [9] advises that if the pulse becomes irregular, 1-50 gr. digitalin should be administered hypodermically every six hours. For more rapid effect in the height of a crisis, strophanthin may be given intravenously.

(9) Transfusion. Many cases are recorded where the transfusion of 300 c.c. of blood to severely hyperthyroid patients, has been followed by immediate improvement.

Rogers [5] claims to have produced and experimented with a so-called anti-thyroid serum in the treatment of

acute thyroid reactions. His results have varied from improvement to marked exacerbation of the symptoms. Very recently he has made a non-coagulable aqueous extract of the thyroid gland which was intended to be used as an antidote in case of overdose of the anti-thyroid serum. Paradoxically, the serum was found to aggravate the symptoms in many cases, while the aqueous extract caused an immediate improvement of the hyperthyroidism. The extract has been given the trade name of "thyroid residue." Rogers claims striking results from its use in many operative and post-operative storms. Obviously, no definite conclusions can be drawn from these experiments at this early stage.

Acute dilatation of the stomach has been not infrequently observed. This complication should be anticipated and promptly treated by gastric lavage.

Under the above treatment marked improvement can usually be expected in 12 to 18 hours.

If the storm has appeared prior to operation, the usual pre-operative measures may then be instituted and continued until the patient's condition is thought to be suitable for some form of surgical intervention. The large majority of these patients who have thus been brought through a crisis, react sufficiently well that some procedure such as bi-polar ligation or a sub-total hemi-thyroidectomy may be performed in the course of ten days to two weeks.

If it has been a post-operative storm, we revert gradually to the usual post-operative treatment whenever the severe symptoms abate.

CONCLUSIONS.

1. Thyroid storms show various degrees of severe hyperthyroidism.

2. Marked metabolic disturbances develop as the storm progresses.

3. Moderate post-operative reactions are fairly common.

4. Severe storms should be relatively rare.

5. They are prevented by careful attention to the principles of the modern surgical management of hyperthyroidism.

6. They demand prompt and adequate medical treatment.

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The Treatment of Some of the Common Fractures.

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The object of this paper is to present a brief outline of the treatment of fractures as taught in this clinic. Anyone who has tried to study the treatment of fractures in the various text books of surgery will realize that there is a great diversity of opinion as to the methods employed. While all these methods probably produce satisfactory results, the treatment outlined below is the one generally accepted in the hospitals in this city. No mention is made of the signs and symptoms of the fractures, as these can be obtained from any text-book; but the displacement of the fragments is given, as this will help in the understanding of the methods used to correct the deformity.

The description of the treatment of any fracture can of course refer only to an average case, and the methods used must be altered to suit individual cases. This is particularly true with regard to the length of time of immobilization; as the time required to produce solid bony union depends on many factors, and the fracture should always be immobilized until there is clinical evidence of solid union. The after treatment of fractures is also very important and while this will not be discussed in this paper, it may be taken as a general rule that the movement of joints adjacent to the fracture should be started as early as possible, removing and replacing splints daily if necessary. I will now consider the various fractures separately.

MANDIBLE:

Position of fracture(1) Just in front of mental foramen; (2) Behind angle of jaw; (3) Coronoid process (rare); (4) Condyle (rare).

Displacement.

Of (1) and (2) Anterior fragment downward due to myelohyoid, digastricus, geneoglossus, geneohyoideus. Posterior fragment upwards due to masseter, temporals, pterygoids.

(3) Of condyle—forwards; (4) of coronoid—upwards.

Treatment.

If there is only slight displacement and the fragments show no tendency to separate, a four-tailed bandage or moulded leather splint may be used to exert pressure backwards and upwards. This should be applied for four weeks followed by limited movement for a few weeks.

If the fragments are separated, other means must be adopted, and in suitable cases, particularly where the fracture is in front of the mental foramen, simple interdental wiring is the standard treatment. The following description of the method is taken from Scudder. In applying and tightening the wire the aid of a dentist should always be obtained. The wire should be No. 24 soft iron wire, annealed copper wire 22 G., or brass wire 24 G. Four teeth are selected on each side of the jaw. One wire should be placed at least one tooth distant from the line of fracture so as not to loosen the teeth adjacent to the fracture. Have the wires about 45 cm. long and wire each tooth separately, twisting the wire always to the right and as tightly as possible around each tooth. Now bring the wires from the upper and lower teeth together in pairs, but diagonally, with gentle but firm pressure, until the fragments are brought into apposition and the teeth approximately into alignment. Then twist all the wires tightly, cut off the projecting surplus of the wire, and cover the ends with softened gutta percha.

All food must be liquid and the mouth must be carefully cleaned after

feeding, and every four hours. After removal of the wire, usually at the end of four weeks, the patient must be careful not to subject his jaw to any violence for some weeks.

RIBS:

Site—Usually occurs in the fifth to eighth rib at the angle.

Treatment.

Strap the affected side firmly with adhesive 1½ to 2 inches wide, which extends beyond the midline in front and back and which is applied during forcible expiration. Overlap the adhesive. Immobilize 3 weeks.

CLAVICLE:

(1) Greater convexity — usually near middle of clavicle between rhomboid and coracoclavicular ligaments. Fracture runs backward and inwards. The inner fragment is raised by the sternocleidomastoid, while the outer fragment is depressed, tilted down and approximated to the mid-line by the weight of the arm.

(2) Between conoid and trapezoid ligament—slight displacement.

(3) At acromial end external to trapezoid—the outer end is dragged downwards and forwards.

(4) At sternal end (rare).

Treatment.

(1) Rest in bed for 3 weeks with sand bag between shoulders and arm bandaged to side. If union isn't firm keep in bed one week longer. This method gives the least callus formation and is especially applicable to women where a good cosmetic result is important.

(2) Sayre's Method. Immobilize three weeks or longer.

(3) Cross Method. The centre piece of a wooden cross is securely fixed over the spine by means of adhesive strips passing around the body and the shoulders are strapped to the cross piece, the arm on the affected side held in a sling. Immobilize three weeks.

HUMERUS.

(1) Anatomical Neck.

Displacement—the head may be impacted into the lower fragment. If completely separated the shaft usually lies in front. The treatment is the same as for fractures of the surgical neck.

(2) Surgical Neck.

Displacement—Upper fragment rotated out by the infraspinatus. Lower fragment upwards by deltoid, biceps and triceps, and inwards by pectoralis major, teres major, latissimus dorsi. Impaction may occur, the lower fragment being driven into the upper.

Correct the deformity and set in plaster with the arm externally rotated, abducted to about 60°, with the scapula hanging free and the point of the elbow in the coronal section of the body. Leave up in this position for six weeks.

(3) Shaft.

Displacement—(1) Below pectoral insertion and above deltoid—upper fragment inwards, lower fragment upward and outwards. (2) Below deltoid insertion—Upper fragment outwards, lower fragment upwards in front of, or behind upper.

Treatment.

The deformity is reduced by traction on the forearm with the elbow bent to a right angle, and manipulation. Set the arm in a position which will maintain alignment, preferably in abduction. If there is no tendency for the displacement to recur, the simplest method of fixation is an external plaster of Paris splint reaching from above shoulder to below elbow and coming sufficiently around the arm to prevent lateral movement. If, however, as often happens, alignment cannot be maintained by this means, a wire splint devised by Dr. George Wilson is used. A plaster jacket is put on the patient and the ends of the wire splint firmly anchored in the plaster.

The arm is abducted to a right angle and the elbow is bent to a right angle. One wire of the splint is above the arm and the other below, and the splint projects several inches beyond the elbow, turns at a right angle, and is continued beyond the fingers. Extension can be applied to the arm by means of adhesive plaster, which can be fastened securely to the splint. Immobilize for eight weeks.

(4) Transverse Supracondyloid.

Displacement—(1) Line of fracture is from before, backwards and upwards. (2) The whole lower fragment with the ulna is carried backwards and upwards from the direction of the force. Occasionally it is carried forward.

Treatment.

Reduce under an anaesthetic by flexing forearm and pulling down, at the same time shoving back on the upper fragment. Put the arm up just less than acutely flexed. When in this position there should be no pain and the radial pulse must be felt. Maintain the flexion by means of adhesive plaster and a sling. At the end of 2 weeks loosen the plaster one inch. Then gradually loosen it until at the end of 4 weeks the elbow is only flexed to a right angle and the plaster may be discarded.

ULNA.

(1) Olecranon:

Displacement—backwards and upwards due to triceps.

Treatment—Open reduction, holding the fragments in place by means of silver wire, kangaroo tendon, or a boiled bone screw.

(2) Shaft.

Displacement—Lower fragment towards radius (pronator quadratus) upper fragment forward (brachialis).

Treatment.—Place the forearm midway between pronation and supination, reduce the deformity and immobilize with anterior and posterior splints for 4 weeks.

RADIUS.

(A) Neck.

Displacement — Lower fragment forwards and upwards.

Treatment—Forearm fully flexed and completely supinated. Immobilize for 4 weeks with early movements.

(B) Shaft.

Displacement.—(1) Above pronator teres insertion the lower fragment is fully pronated and drawn inwards by both pronators, and the upper fragment is supinated by the biceps and supinator.

(2) Below pronator teres insertion the upper fragment is drawn forwards and inwards (biceps and pronator) in a position between supination and pronation and the lower fragment is drawn inwards by pronator quadratus.

Treatment.—Set (1) in complete supination, and (2) midway between supination and pronation and immobilize 4 weeks.

(C) Colles.

Displacement of lower fragment. (1) Upwards towards elbow. (2) backwards by the direction of the force. (3) Rotated around ulnar styloid as centre so that outer part is more displaced than inner. (4) Rotated backwards around line of fracture, so that the back of the fragment is more displaced than the front.

Treatment.—Disempact by standing in front of the patient and grasping his hand as though you were shaking hands with him, using your right hand for fractures on right side, and left hand for those on left side. With counter extension made against the flexed elbow forcibly extend and adduct the hand, and this will usually suffice to disempact it. The fragments are molded into place by a little manipulation, and held in place by anterior and posterior splints extending from knuckles to elbow. The hand is usually set in the cocked-up position, but if necessary to obtain alignment it may be flexed for 10 days and then set up. Immobilize

for 3 weeks, being sure to move fingers and thumb from the beginning.

FEMUR.

(1) Neck.

Displacement. — Rotated outwards by its weight and drawn up by the hamstrings and quadriceps. The capsule prevents much shortening, which is usually about 1 inch.

Treatment.—Internally rotate leg. Then abduct to 45° and pull until the length is restored as shown by measurement and comparison with the other leg. Have patella pointing up when the plaster is applied, and put on spica, from ball of foot to just below breasts, and leave on for 12 weeks. At the end of one month split plaster to knee to allow flexion of knee.

(2) Shaft.—Upper, Middle, and Lower third.

Displacement.—The lower fragment is always drawn up by thigh muscles and everted by the weight of the leg. In the upper third the upper fragment is flexed by the psoas and the lower is adducted. In the middle third there is a tendency to backward sagging, the lower fragment being drawn up either in front or behind the upper. In the lower third the lower fragment is flexed at knee by gastrocnemus.

Treatment. (1) Upper or Middle Third.—Use a Thomas splint and Buck's extension, keeping the knee and leg straight and putting the plaster 4 inches above the site of fracture. Use a bias cut flannel bandage to hold plaster on leg. The extension should be applied within 24 hours and sufficient weight should be used to restore the length of the leg—up to 40 lbs., if necessary. When length is restored, reduce the weight; and when union occurs reduce the weight to one-half and leave on for 2 or 3 weeks longer. The foot of the bed is raised for counter extension. Immobilize for 8 weeks and then prevent weight-bearing for another month by means of a Thomas walking caliper. In transverse fractures of the middle of shaft, if length is restored and corners are touching the result is as good as if the fragments were on end.

For fractures of the lower third use a Thomas splint bent at knee, and Buck's extension; or if this isn't satisfactory, use ice-tong calipers. The knee should be flexed 60° on the thigh.

PATELLA.

Displacement.—Usually marked separation of fragments.

Treatment.—Open operation. Remove all clots and fibrous tags from between the fragments. Sesure the fragments with silver wire, chromic catgut, or kangaroo tendon. Extend knee and set in plaster for 6 weeks if wire is used. Begin movements after 1 month, but no weight bearing. Protect from injury and full extension for several months.

TIBIA AND FIBULA.

Displacement.—From indirect violence the tibia usually breaks at the junction of the middle and lower thirds, the fibula higher up. The fracture of the tibia is oblique, downwards, forwards and inwards. The lower fragment is drawn up and rotated outwards. The upper fragment often pierces the skin.

Treatment.—Flex the knee to a right angle and place foot in a position of rest. Apply plaster to the leg, and foot, above and below the fracture, running the plaster above the knee. Let the plaster harden. Then set the fracture and while it is held in position, apply plaster in the interval between the other two plasters. This method prevents the fragments slipping out of place. Leave in plaster 3½ weeks, then straighten knee and reapply plaster for 3 weeks more. Then remove plaster, but allow no weight bearing for another two weeks. There are more cases of non-union of tibia than any other bone, and if this occurs open reduction with a bone graft is necessary.

POTT'S FRACTURE.

Displacement.—The foot is displaced and rotated. (1) Outwards (commonest); (2) inwards; (3) backwards.

Treatment.—Flex the knee and pull the foot inwards as far as it will go, and forwards as far as possible. Make sure the centre of the body of the talus corresponds to the centre of tibia. Set the foot at right angles and adduct as much as possible and apply plaster for 4 weeks, then remove and start movements and allow weight bearing in 2 weeks, with a ¼ inch lift on the inside of the sole of the shoe.

The Oldest Surgical Treatise.

By O. E. A. STEPHENS, 3T4.

When did man first begin to be aware of the mysteries of his own body, study his structure and investigate the nature of the extraordinary process which it carries on? It has been said that astronomy is the oldest of sciences, and it is true that astronomy developed astonishingly early; but behind the beginnings of astronomy, mathematics and medicine, there were long stages of practical human experience which were gradually transformed into science, and it would be difficult to determine with certainty which was the earliest of the group.

If we could decide the question on the basis of surviving treatises, then medicine and mathematics could claim priority, for we possess Egyptian treatises in these fields which are considerably older than any surviving astronomical discussion; but such a decision is impossible, for all three of these sciences are much older than any surviving treatises.

As far back as the middle of the 28th century B.C., we find that Pharaoh Neferirkere had in his possession a "case of writings" which was a case of some kind for carrying medical treatises. Unhappily, none of the actual rolls from that royal "case of writings" has survived to our day. Nevertheless there is plenty of evidence of the advanced knowledge of medicine and surgery in the first great age of Egyptian civilization, which we call the Pyramid Age (about 3000 to 2500 B.C.).

The court of the Pharoah offered opportunity for medical specialization even at this remote period. A tombstone recently found in the vast Gizen cemetery belonged to a certain Iry, who was not only chief of the palace medical corps, but also served the Pharoah as "Palace Eye-Physician" and furthermore as "Palace Stomach-Bowel-Physician" in which capacity he bore the titles, "One understanding the internal fluids," and "Guardian of the Tnus," showing that he was particularly versed in internal medicine and that he specialized in diseases of the digestive organs.

As far back as the period beginning 3000 B.C., therefore, the extraordinary civilization of the Nile Valley already possessed the practice of surgery and had likewise produced specializing physicians.

The earliest physician whose name has come down to us, was the illustrious Imhotep, who lived not long after 3000, that is the 30th century B.C. He served the Pharoah Zoser, whose Step Pyramid at Sakkara in the desert behind ancient Memphis is familiar to all tourists in Egypt.

Imhotep was an illustrious physician whose name survived in later ages to become the Asclepios of the Greeks, the Aesculapius of the Romans. No writings of this earliest known medico have

descended to us; and not a single medical roll from that royal "case of writings" has survived to our day.

That original autograph manuscript left by the author himself undoubtedly perished ages ago, but copies of it were still in existence a thousand years after the author's death. All of these long ago disappeared, with the exception of one, which was made in the 17th century, B.C. This one, in so far as we know, the only surviving copy, is in the collection of the New York Historical Society. It is the oldest known surgical treatise, and at the same time the earliest surviving scientific discussion.

In physical form this extraordinary document is a strip of papyrus a little over 15¼ feet long, and about 13 inches wide. At least a trifle over a foot and possibly much more, has been lost at the beginning of the strip, so that it was originally not less than 16 feet, 4 inches in length. When not in use the papyrus was rolled up, not unlike a roll of narrow wall paper.

The scribe of the 17th century B.C., taking up the clean unwritten roll, began copying at the right end of the papyrus. In columns over 11 inches high and about 8 inches in width, his copy marched on, column after column from the right end toward the left end of his new roll.

The older roll set up before him, from which he was copying, may already have been centuries old. The venerable surgical treatise which it bore, however, was then already something like a thousand years old, and of course contained old words and phrases which were no longer wholly intelligible. Centuries earlier some one had written out a series of explanations and discussions of these obscure words and phrases at the close of each case discussed, so that each case was followed by a little dictionary of its difficult terms.

He had copdied at least 18 columns (we do not know how many columns there were in the lost introduction) containing some 400 lines, and had reached the bottom of a column when, pausing in the widdle of a sentence, in the middle of a word, he lifted his pen and ceased writing, never to complete his work.

His copying had carried him from the human head systematically downward through the neck to the thorax, and had begun the first case on the spinal colum when he stopped. The copy presents case after case through successive, carefully arranged discussions, forming a series of 48 cases.

The surgical treatise itself probably served as a reference book consulted daily by the practising physician, while at the same time it partook of the nature both of the medical lecturer's outline and the student's notebook; for the ancient medical student will have roughly reproduced the form and content of his instructor's lecture notes and may afterward continued to employ his note book as a reference hand book.

The papyrus nowhere hints at the name or station of the author who produced this earliest-known investigation of human anatomy, physiology, and pathology. It is evident from his work that our ancient surgeon was a man of observant and discerning mind, with a wide outlook upon the life of his time. The terms he uses convey the impression of a man actually involved in the process of building up a terminology in a field of observation not yet possessing a fund of current terms.

He draws his items of comparison and his descriptive terms from nature, from the mechanical arts, from architecture and from many sides of daily experience. In describing the articulation of the human mandible with the skull, for instance, he likens the fork at the head of the ramus where the mandible hangs pivoted, to the claw of a two-toed bird clasping the temporal bone—a very apt comparison.

The writer was already acquainted

with the brain, and the word "brain" appears in the treatise for the first time in human speech, in so far as early records are preserved to us. He had observed that the brain is in intimate connection with the control of the limbs, and he had already noticed the localization of function of the brain. He charges his readers to note the side of the skull on which an injury is found, and to correlate it with the side on which paralysis of the lower limbs occur. He was aware of a cardiac system, with the heart as its centre, and he knew that the action of the heart extends to the remotest extremities, but he does not indicate a knowledge of the circulation of the blood, although he was without a doubt close to that observation. His directions for setting dislocated and fractured bones show a good understanding of the proper physical manipulation. Many of these instructions were closely reproduced in an illustrated edition of the essay on the joints written by Hippocrates over two thousand years later than our Egyptian surgical treatise.

It is quite evident that the work and writings of such Egyptian medical men could not have remained unknown to the great Greek physicians, who carried on their investigations in Alexandria after 300 B.C., side by side with the Egyptian medical schools. Our unknown Egyptian surgeon and his colleagues reaching back to Imhotep of 5,000 years ago, were therefore the scientific ancestors of modern medical scientists who have probably been accustomed to regard their scientific ancentry as reaching no further than the Greeks. The modern physician, however, is entitled to consider himself as a descendant of a venerable line which may be traced back to the 30th century B.C., and this can not be said of any other modern scientist.

Sodium Amytal.

By W. A. HAWK, Med., 3T0.

Quite recently at the Toronto General Hospital we have had several cases which were anaesthetized by sodium amytal, it has been used as an analgesic during labour, consequently our curiosity was piqued as to the nature of the drug. It is with a view to presenting some of the facts known about this drug that this paper was written.

Amytal is one of the barbital compounds — Sod Iso-amyl-ethyl-barbiturate, and during the past six years this has been used extensively as a general anaesthetic in animal experiments. It may be prepared in a pure anhydrous form and, if sealed in a glass vessel, is perfectly stable. Highly purified preparations adjusted to a p H between 9.5 and 9.8 give the maximum anaesthesia in dogs with the minimum toxicity. A lowering of the p H to the neighbourhood of 9.2 shows a striking loss in anaesthetic value and a marked increase in toxicity, producing Cheyne Stokes breathing and other toxic effects in the respiratory centre. It might be thought that a similar condition would occur in man when the p H fell to 7.4 in the blood. However, there is no trace of such occurring. It might be well to note that too rapid injection may affect the respiratory centre.

In the animal, 40-50 mgm, when

slowly injected, intravenously in a dog will cause complete loss of consciousness in three or four minutes, sufficiently deep surgical anaesthesia in about 20 minutes. The anaesthesia lasts from 1 to 3 hours depending on the dosage, following this is a somewhat prolonged period of post-operative sleep from which the dog may be aroused. As an antidote and as a means of counteracting this sleep we may inject ephedrine sulphate followed by caffeine soda benzoate. However, during the anaesthesia, it has been noted that amytal depresses the circulation and may cause temporary paralysis of the vagus.

Coming to the practical side of this paper, sodium amytal has given excellent results in the induction of general anaesthesia in man. A dosage of 10 to 15 mgm. per kilo. has generally produced adequate anaesthesia for satisfactory manipulation in minor surgery. 20 to 25 mgm per kilo. have produced complete surgical anaesthesia in cases where other general or local anaesthetics were counter-indicated or inconvenient to administer; wherever possible most anaesthetists are using it in conjunction with general or local anaesthetics. The intravenous injection of from 12 to 20 mgm per kilo. has made it possible in N_2O and O_2 or ether anaesthesia to reduce the supplementary anaesthetic required to 20-25% of the quantity usually employed. That is, we might use N_2O, 25% for complete relaxation and surgical anaesthesia.

Recent observers have noticed that preliminary amytalization with a local anaesthetic has produced more profound anaesthesia and greater relaxation. In addition, it may diminish the toxic and convulsive effects of certain of these anaesthetics—chiefly procaine.

It is of the utmost importance that the preparation should be in perfect solution before being injected and the rate of admission intravenously should not be greater than 1cc per minute. Sleep as a rule commences within 3-5 minutes, and surgical anaesthesia with complete relaxation of the muscles and loss of the common reflexes except the pharynegeal, sphincters, etc., occurs within 15-20 minutes after starting the injection. The respirations are those of a patient in deep sleep, more often increased than decreased in rate, somewhat diminished in amplitude but regular. The blood pressure may fall 15-30 mm. during the time of injection but rapidly regains the former level.

Surgical anaesthesia lasts for a period of 1-3 hours, dependent in some measure upon the dose employed. After maximal doses the patient may sleep from 3-10 hours. Nausea, retching and vomiting have not been observed in any of these cases. In those cases where it is desired that the patient should wake somewhat sooner and fully alert mentally, 25-50 mgm. of ephedrine sulphate injected intramuscularly followed by 0.65-1.0 gm. of sodium caffeine benzoate, one-half to two hours after the operation, has the desired effect.

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